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FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, CANCERLIT, BIOTECHDS' ENTERED AT  
14:53:09 ON 04 JUN 2003

L1 3099 S 100K OR NUCLEOTID? 9###  
L2 109595 S ADENOVIR?  
L3 246 S L2 AND L1  
L4 82 DUP REM L3 (164 DUPLICATES REMOVED)  
L5 2315221 S DELE? OR REMOV?  
L6 632978 S DEFICIENT OR LACKING  
L7 2890079 S L6 OR L5  
L8 16 S L7 AND L4

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L3 with l2 22

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L4

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DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ

<u>L4</u>	L3 with l2	22	<u>L4</u>
<u>L3</u>	adenovi\$	22259	<u>L3</u>
<u>L2</u>	100K or nucleotide 9??? or nucleotides 9???	6045	<u>L2</u>
<u>L1</u>	100K or nucleotide 9???	6045	<u>L1</u>

END OF SEARCH HISTORY

L8 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2003 ACS

AN 2000:161478 CAPLUS

DN 132:204060

TI **Adenoviruses deleted in the IVa2, 100K**  
and/or preterminal protein sequences

IN Amalfitano, Andrea; Chen, Yuan Tsong; Hu, Huimin

PA Duke University, USA

SO PCT Int. Appl., 156 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 20000012740	A2	20000309	WO 1999-US19540	19990827
	WO 20000012740	A3	20001123		
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2340276	AA	20000309	CA 1999-2340276	19990827
	AU 9956942	A1	20000321	AU 1999-56942	19990827
	EP 1108049	A2	20010620	EP 1999-943952	19990827
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	US 6328958	B1	20011211	US 1999-384749	19990827
	JP 2002528056	T2	20020903	JP 2000-567725	19990827
PRAI	US 1998-145742P	P	19980828		
	WO 1999-US19540	W	19990827		
AB	The present invention provides <b>deleted adenovirus</b> vectors. The inventive <b>adenovirus</b> vectors carry one or more <b>deletions</b> in the IVa2, 100K, polymerase and/or preterminal protein sequences of the <b>adenovirus</b> genome. In the human <b>adenovirus</b> serotype 5 genomes, such <b>deletions</b> are at nucleotide positions 4830-5766, 24,990-25,687, and/or 7274-7991. The <b>adenoviruses</b> may addnl. contain other <b>deletions</b> , mutations or other modifications as well. In particular preferred embodiments, the <b>adenovirus</b> genome is multiply <b>deleted</b> , i.e., carries 2 or more <b>deletions</b> therein. The <b>deleted adenoviruses</b> of the invention are "propagation-defective" in that the virus cannot replicate and produce new virions in the absence of complementing function(s). Preferred <b>adenovirus</b> vectors of the invention carry a heterologous nucleotide sequence encoding a protein or peptide assoccd. with a metabolic disorder, more preferably a protein or peptide assoccd. with a lysosomal or glycogen storage disease, most preferably, a lysosomal acid .alpha.-glucosidase. The <b>deleted adenovirus</b> vectors advantageously have an increased carrying capacity for heterologous nucleotide sequences, demonstrate lower levels of viral protein expression, induce fewer host immune responses, and/or exhibit increased stability and prolonged transgene expression when introduced into target cells.				

L8 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2003 ACS  
AN 2002:946147 CAPLUS

DN 138:34131

TI Helper-virus independent replicating **adenovirus** vectors with  
**100K** or E1b gene **deletion** for gene therapy

IN Amalfitano, Andrea; Hodges, Bradley L.

PA Duke University, USA; Koeberl, Dwight D.

SO PCT Int. Appl., 75 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI WO 2002098466 A1 20021212 WO 2002-US17070 20020531

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,  
CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,  
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,  
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,  
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,  
UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,  
TJ, TM  
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,  
CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,  
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-295914P P 20010604

AB The present invention provides replicating [100K-]  
**adenovirus** vectors that have an impairment in **100K**  
activity. In particular preferred embodiments, the impairment is the  
result of a **deletion** in the **100K** coding region of the  
**adenovirus** vector genome. It is further preferred that the  
**adenovirus** produces the E1 gene products. In an alternate  
embodiment, the **adenovirus** produces the Ela gene products, but  
has an impairment in the E1b coding region, such that replication of the  
virus is limited to p53- cells. Also described are methods of making and  
administering the inventive **adenovirus** vectors to a cell or to a  
subject. Further provided is use of the inventive [100K-] Ad  
vectors as a helper virus for the prodn. of vector stocks of adeno-assocd.  
virus.

L8 ANSWER 7 OF 16 MEDLINE  
AN 83303837 MEDLINE  
DN 83303837 PubMed ID: 6612996  
TI Analysis of Ad5 hexon and **100K** ts mutants using conformation-specific monoclonal antibodies.  
AU Cepko C L; Sharp P A  
NC NIH-P01-CA14051 (NCI)  
P01-CA26717 (NCI)  
SO VIROLOGY, (1983 Aug) 129 (1) 137-54.  
Journal code: 0110674. ISSN: 0042-6822.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 198310  
ED Entered STN: 19900319  
Last Updated on STN: 19970203  
Entered Medline: 19831008  
AB **Adenovirus** type 5 ts mutants **deficient** in hexon metabolism were investigated using conformation-specific monoclonal antibodies directed against hexon capsomeres and the viral **100K** protein. The ts mutants map either in the hexon structural gene or in the gene encoding the **100K** protein, a major, late nonstructural protein. All of the mutants examined (ts1, ts2, ts3, ts4, ts17, and ts20 of J. F. Williams, M. Gharpure, S. Ustacelebi, and S. McDonald (1971). J. Gen. Virol. 11, 95-101) were unable to produce the capsomeric form of hexon (a trimer of three hexon monomers) at the nonpermissive temperature. However, all of the mutants retained the ability to produce a complex of **100K** and hexon which has been demonstrated to play a major role in the assembly of hexon trimers. The mutants accumulated nontrimerized hexon in this ts complex in the perinuclear region of the cell. Several of the mutants (ts1, ts2, ts3) were found to successfully assemble hexon synthesized at the nonpermissive temperature upon shift down to the permissive temperature, even in the presence of a protein synthesis inhibitor. The mutant, ts2, which maps in the hexon structural gene, was found to be dependent on protein synthesis for transport of hexon trimers into the nucleus during temperature shift down, while the **100K** ts mutants, ts1 and ts3, were independent of protein synthesis for both hexon assembly and transport.

L8 ANSWER 1 OF 16 MEDLINE  
AN 2001320301 MEDLINE  
DN 21286721 PubMed ID: 11390592  
TI **Adenovirus** vectors with the **100K** gene **deleted**  
and their potential for multiple gene therapy applications.  
AU Hodges B L; Evans H K; Everett R S; Ding E Y; Serra D; Amalfitano A  
CS Department of Pediatrics, Division of Medical Genetics, Duke University  
Medical Center, Durham, NC 27710, USA.  
NC DK52925 (NIDDK)  
SO JOURNAL OF VIROLOGY, (2001 Jul) 75 (13) 5913-20.  
Journal code: 0113724. ISSN: 0022-538X.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 200106  
ED Entered STN: 20010702  
Last Updated on STN: 20010702  
Entered Medline: 20010628  
AB The **100K** protein has a number of critical roles vital for  
successful completion of the late phases of the **adenovirus** (Ad)  
life cycle. We hypothesized that the introduction of **deletions**  
within the **100K** gene would allow for the production of a series  
of new classes of Ad vector, including one that is replication competent  
but blocked in the ability to carry out many late-phase Ad functions.  
Such a vector would have potential for several gene therapy applications,  
based upon its ability to increase the copy number of the transgene  
encoded by the vector (via genome replication) while decreasing the side  
effects associated with Ad late gene expression. To efficiently produce  
**100K-deleted** Ad ([**100K-**]Ad) vectors, an E1-  
and **100K**-complementing cell line (K-16) was successfully  
isolated. Transfection of an [E1-,**100K-**]Ad vector genome into  
the K-16 cells readily yielded high titers of the vector. After infection  
of noncomplementing cells, we demonstrated that [**100K-**]Ad  
vectors have a significantly decreased ability to express several Ad late  
genes. Additionally, if the E1 gene was present in the infected  
noncomplementing cells, [**100K-**]Ad vectors were capable of  
replicating their genomes to high copy number, but were significantly  
blocked in their ability to efficiently encapsidate the replicated  
genomes. Injection of an [E1-,**100K-**]Ad vector *in vivo* also  
correlated with significantly decreased hepatotoxicity, as well as  
prolonged vector persistence. In summary, the unique properties of [  
**100K-**]Ad vectors suggest that they may have utility in a variety  
of gene therapy applications.

**WEST** [Generate Collection](#) 

L4: Entry 3 of 22

File: USPT

Dec 10, 2002

DOCUMENT-IDENTIFIER: US 6492343 B1  
TITLE: Porcine adenovirus type 3 genome

Other Reference Publication (3):

McCoy et al. Nucleotide and Amino Acid Sequence Analysis of the 100K Protein of a Serotype 3 Porcine Adenovirus. DNA Sequence-The Journal of Sequencing and Mapping, vol. 8, pp. 59-61, 1997.\*